Scientific Analysis and Health Impact of Propane Levels in:

Ambient Air Data of Erie Colorado
Presented By NOAA Earth System Research Laboratory
February 21, 2012 Town of Erie Study Session

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QEPA Commitment to Scientific Integrity

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Declaration of Conflict of Interest

Authors Wright and Havics have previously performed significant work for the Oil &Gas Industry. They have also been invited speakers for EPA on technical and health implications of Fracing.

A Note to the Reader

This report is in response to the increased community concern surrounding the reported levels of propane and whether there is sufficient evidence in the data from the NOAA Presentation to suggest increased health effects from oil & gas activities in the Erie area. It is not intended to address the longer term policy of permitting oil & gas activities, other than to convey the value of this data by itself. It should be noted that we have critically reviewed the NOAA presentation from a scientific standpoint. In doing so, it is both easier and more expedient to focus on the weaknesses as opposed to the strengths in data under review, as these are essential in determining the level of reliability and relevance. Science, in itself, is a process of critically testing a hypothesis, thus the approach contained herein. Also, we have addressed the perception of the general public, correctly or incorrectly formed, as well as content and context of the message delivered, intentional or unintentional; for it is the perception of the people that is important in terms of both environmental, health and safety policy and risk communication.

EXECUTIVE SUMMARY

A scientific analysis of the data presented by Dr. Steven Brown on February 21st was conducted by QEPA. We considered two aspects, the quality and context of the data in regards to its technical merit, and the relevance of any health risk associated with the level measured. For the first part, the limitations, relevance and reliability of the data were critically reviewed.
The findings are as follows:

- The small seasonal sampling period and limited data set that was used is not adequate to assess conditions.
- The data is wind direction dependent and not accounted for here.
- Data is qualitatively but not statistically reviewed, which limits the authors ability to draw appropriate conclusions.
- The emissions inventory does not directly reflect air data.
- The data, as presented are limited and not reliable in determining the source of the VOCs.

QEPA was also tasked with assessing whether the [1]increased levels of propane presented by Dr. Brown would present health concerns to the residents of Erie. A search was conducted to determine available human health guidelines for propane. This search included a review of Federal Regulatory Agency documents. QEPA also did a limited literature review for studies of propane exposure and poisoning.
The findings are as follows:

- Health guidelines for propane exist for occupational exposures but not for general human health.
- A limited review of studies for instances of propane poisoning revealed mainly two types of scenarios, accidental exposure in enclosed environments and accidental overdose during recreational intentional self-exposure similar to "huffing" or taking drugs.
- Studies were not found where propane levels in the ambient air were confirmed as a cause of health effects.
- The levels of Propane presented in the NOAA study are a 1000-fold or more below those considered to be of health concern.

BACKGROUND/STATEMENT OF ISSUE

Purpose of NOAA study
The purpose of the study was to assess Volatile Organic Chemicals, (VOCs) components as part of overall air quality in the region.

Scope of Work on the NOAA Study
The basic scope of work for the NOAA study was to evaluate halogens, aerosols in general, nitrogen oxides (NOx), and the mix of these in the generation of air pollution. The scope was expanded to include determination of aerosol composition and sources within the Denver Brown Cloud" and emissions from the oil and gas industry. It is this last add-on regarding oil & gas that is the focus here. The sampling regarding this portion focused on Volatile Organic Compounds (VOCs) including propane.

[1] The NOAA presentation does not state that there was an increase. In a communication with the author he indicated that he did not state there was an increase, but may have stated levels of propane were elevated compared to data from Los Angeles, CA and Houston, TX.

ANALYSIS OF DATA USED

Methodology of the NOAA Study

The NOAA used a single sampling location, the BOA tower. At that location, they collected samples from different heights during the night and day. They collected wind direction data to correlate the location from which the VOCs might be coming from. Samples were analyzed by Gas Chromatography (GC) Mass Spectroscopy (MS). It is not stated in the NOAA presentation, but we are aware of other NOAA processes that use rigid and proper QA/QC procedures. So the quality of the data is expected to be good. This presentation used a small February through March data set to assess conditions. It is not stated in the NOAA presentation why this seasonal limited period was used.

General Context, Content, and Presentation of Data of NOAA Study

The presentation provides concentrations relative to one another for a variety of alkanes (mainly simple gases like Butane, Propane, and Hexane), aromatics (like Benzene, and Toluene that are well associated with gasoline and certain oil and gas sources) and Alkenes (higher order compounds associated with more processes; soil & gas sources as well as other conventional industry emissions). Data was presented in comparisons to Propane, because Propane is presumed to be a good indicator of oil & gas activities.

The presentation covers wind directionality versus concentration for alkanes and alkenes. It reviews concentration differences by height and concludes that high samples verses ground samples can (and should) be separated because of their difference. They also allocate a large source of alkanes (propane) from local oil and gas activities by qualitative or semi-quantitative comparisons.

The author shows levels of propane averaging over 10 parts per billion (ppb) with peaks up to 115 ppb. They state that some data are more indicative of urban air sources for their study. They conclude that emissions will have longer lifetimes but less ozone production.

Data Limitations and Assumptions of the NOAA Study

There are several limitations noted in the presentation:

- *Data is Wind direction dependent.*
  Although the data was collected to allow evaluation of the VOCs by wind direction, only a lumped group is used to determine what is the source of the pattern of chemical. Since the data is grouped without respect to wind direction, the source(s) could be from Denver, Boulder, the DJ Basin, and so on. There is no way to tell for sure.
- *Results are clearly height dependent, and weather dependent.*
  Because the concentrations are height dependent and they have not addressed from what location the sources start from, there is no sure way to know how far the source traveled before getting to that height. Thus one cannot say for sure whether it was near or far. In addition, because the weather-wind pattern determines the flow of air (boundary layer development and dispersion), without accounting for the weather, the determination of the source strength and location are also suspect.
- *They used February-March 2011 data only (seasonally and limited in number), the results of which also contradicts some 2008 data.* Based on other data for the area, the mixing values change by season, possibly affected by temperature. The prevalence of wind direction also changes by season. Because of the above limitations and because the data is different from 2008 data (see the Petron study[1]), the reliability for drawing broad conclusions is weak. There are some other aspects that limit the usability of the data for the Erie area. The data is from a single location, with no upwind or downwind comparisons. The distance of BOA tower used for the sampling is away from actual homes in Erie and has not been calibrated to the Erie location.

The NOAA author assumes:

- *Limited data is sufficient to make these conclusions.*
  Because of the weather, wind, height, and seasonal limitations discussed above, the assumption that this data can be used to draw reliable conclusions should have been tempered with language to indicate "preliminary" or "limited", because the data are just that.
- *That the emissions inventory should directly reflect air data (ignoring decay or time to sampling location*).
  The comparison to oil & gas emissions presumes that the sampling location will consistently allow for the capture (by air sampling) of these emissions. This means that at a certain height at a single location one can capture the emissions from a particular source repeatedly. This depends on weather, wind, temperature, and seasonal affects, also being consistent enough or alternatively but using only data that represent controlled subsets of these, which was not done here.
- *That a qualitative evaluation of the data and not a statistical evaluation, is good enough to draw their conclusions.*
  The data were presented as single sets or grouped averages, which is a start. However, they did not use statistical measures of significance such as T-test, F-test, principal component analysis (PCA), or cluster analysis (CA). In order to tell whether something is really different (more than just that caused by inherent variability) requires more comprehensive statistical analysis than was reported here.

Relevance and Reliability of NOAA Data

The only real implication in this presentation is that the source of alkanes is mostly oil & gas related. The data are limited and not reliable for sourcing statements as presented especially since they are different from 2008 and they show height issues as described above.

There is a reference to the Petron study[2] wherein the oil & gas industry is considered the primary source of VOCs, through the correlation of chemicals to propane similar to that used here. It also suggests that the oil &gas industry is underestimating their true emissions and potential impact on the air quality. We note that the Petron study is inherently flawed and contradicts some aspects in this presentation. The Petron Study was primarily done by scientists from the *Cooperative Institute for Research in Environmental Sciences, University of Colorado, and NOAA.*

- They used data from 2008 from the same tower plus a limited set of samples from a mobile van driven around the area.
- They funneled down the data to smaller data sets to evaluate where the hydrocarbon emissions might have come from.
- They used ratios in comparison to propane (such as that used here) and other compounds. They ultimately insinuated a few aspects, as was done here, including that
  a) the source of hydrocarbons is primarily from oil & gas,
  b) the oil & gas emissions are much greater than reported, and
  c) there are ties (correlation) to benzene which is a potential health concern.

Thus, there is a connection here between these studies and their use to imply (without a proper scientific assessment) that oil & gas is having a much greater impact upon air quality than was previously thought. Given this trend, it would be advisable to formally challenge certain aspects of both of these studies.

[2] Pétron, G., et al. (2012), Hydrocarbon emissions characterization in the Colorado Front Range: A pilot study, J. Geophys. Res., 117, D04304, doi:10.1029/2011JD016360

HEALTH IMPACT OF PROPANE LEVELS

QEPA was asked to assess what health concerns (if any) would the elevated levels of propane mentioned in the NOAA study pose to the residents of Erie. There are three items of important note;
1) No health effects are implied or discussed in the NOAA presentation,
2) The author presents levels of propane but never states that they are increased,
3) Discussions on health effects are provided in an effort to provide factual information in areas where there may be public perception of an associated health risk with environmental exposures.

Propane is:

- A non-renewable energy rich gas.
- A liquefied petroleum gas found mixed with natural gas and oil.
- It is separated from natural gas at gas processing plants or from crude oil at refineries.
- It naturally occurs as a gas. At higher pressure or lower temperatures, it becomes a liquid.
- Propane is a clean burning fossil fuel that emits water vapor and carbon dioxide, a greenhouse gas.
- The 2nd largest alternative transportation fuel in use today.
- Also known as dimethyl methane, methyl ethyl methane, n-propane, and Liquefied Petroleum Gas (LPG).
- It is heavier than air. It will disperse into the air if it is released in an open environment, and can pose an explosive risk if it concentrates enough and is ignited.

Major Uses[3]

Homes

- 49 million use to meet some energy need
- 6 million use it as a heating source, patios etc.
- Water heaters, gas grills, cooking, clothes dryers refrigeration, cooling and heating air

Farms

- 865,000 farmers use propane
- Dry crops, ripen fruit, heat water, control weeds
- Heat barns, greenhouses, chicken houses
- Farm equipment operation

Businesses

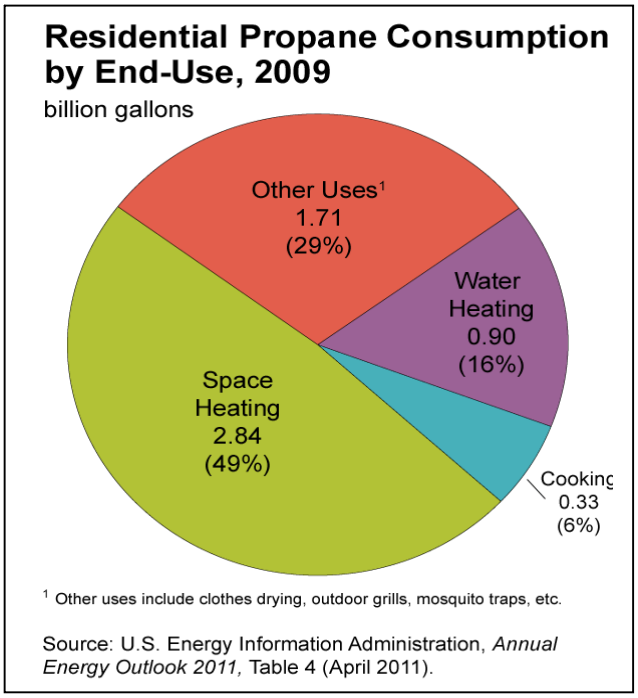
- 1 million use propane as an energy source
- For heating and cooling air, and water
- Refrigerant for foods

Industry

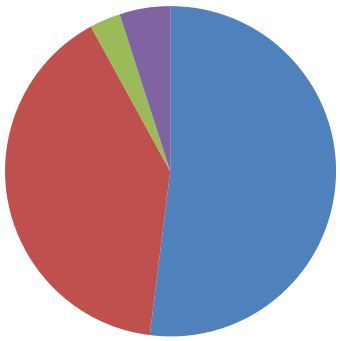
- > 350,000 use propane as fuel
- Soldering, vulcanizing, torches

Transportation

- Taxicabs, school buses, police cars



[3] Energy Information Administration



Propane Consumption 2009

52% Chemical & Industrial

40% Residential & Commercial

3% Transportation

5% Agriculture

Source: Energy Information Administration

COMMUNITY HEALTH CONCERNS

Health concerns noted by QEPA at the February 21st Town of Erie Board of Trustees Study Session and at the February 23 Erie Rising public forum were:

- Endocrine Disruption
- Diabetes
- Asthma/ Upper Respiratory Issues

Excluded Community Concerns
Endocrine disruption *was excluded* from further review as propane does not exhibit hormone like properties. The theory of endocrine disruption is that natural and man-made substances that exhibit hormone-like properties in the laboratory can also affect wildlife populations, thus speculating that humans may be at risk of "hormone disruption".

Diabetes *was excluded* from further review as propane has not been associated with this disease. Factors that lend themselves to the onset of diabetes are: age, genetics, low physical activity, obesity, and diet.

Health Issue of Community Concern - Asthma
Of the three areas of concern, asthma/ upper respiratory issues was selected for assessment of health effects relative to the presented propane levels.

Review Approach
A search of federal databases for studies relating to propane and asthma was conducted. A review of Weld County health information was also done.

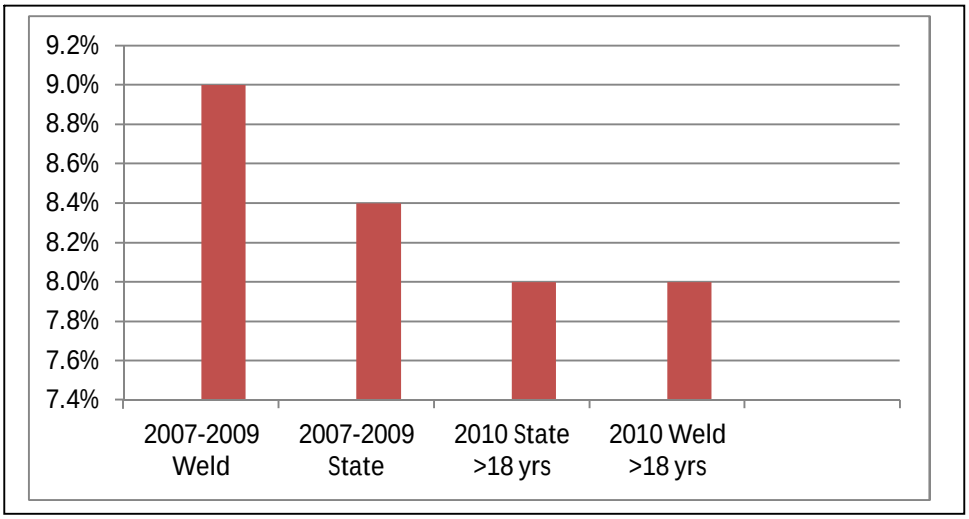
What is Asthma?
It is a chronic disease of the lungs. It is characterized with episodes of wheezing, breathlessness, chest tightness, and night time and early morning coughing.[4] In most case it is not known what causes it. It is known that if someone in your family has asthma, you are more likely to get it.

[4] Centers For Disease Control and Prevention - Basic Information on Asthma www.cdc.gov

What triggers Asthma Attacks?

Asthma symptoms have two main causes of the air passages narrowing. One is due to inflammation and the other, airway constriction, resulting in difficulty breathing. Environmental triggers include: tobacco smoke, air pollutants (ozone), house dust mites, cockroach particles, cat and dog dander, and mold. It is important to note that propane has not been noted to trigger asthma attacks.

Historical County Data - Asthma



Health Information on Asthma- 2012 Weld County Health Status Report[5]

A comparison done by Weld County Health Department on the prevalence of asthma occurrences in Weld County and the state was reviewed. *See Historical County Data – Asthma table above*

> *"…In 2010, the prevalence of asthma in children is 9.4 percent in the U.S. (National Health Interview Survey). According to three years average from 2007-2009 in Weld County, 9 percent of children ages 1-14 years had asthma compared to 8.4 percent of children the same age statewide. In 2010, 8 percent of Weld adults aged 18 years or older reported currently, having asthma which is similar to the 2009 state rate."*

The Weld County Health Department reviews health data for the county and chooses a "Local Issue of Concern" to strategically address annually. Asthma was not chosen by the county health department as an issue of concern. The top two areas of concern were: Nutrition, physical activity, and obesity, and Mental health & substance abuse. The local issues of concern are chosen using the following criteria:

1. Local data shows that many people are either affected or at increased risk of mortality, morbidity, or disability because of the issue,
2. Local capacity assessment indicates that the county has the ability to impact these issues due to the community's readiness and the availability of evidence-based strategies and best practice programming locally, and
3. There are organizations, resources, and local champions that can move the issue forward in terms of public health.

Air quality in Weld County is in attainment with the National Ambient Air Quality Standards for all EPA pollutants except for ozone. The 2012 Health Status Report indicates the following:

> *" the southern two thirds of Weld County is part of the 9 county Denver Metro area*

[5] 2012 Weld County Health Status Report – Community Health Improvement Plan, A Road map for Improving Weld County's Health.

where there are primarily respiratory impacts on human health"

It is important to note that Erie along with Greeley, Evans, and Firestone make up more than 50 percent of the county's growth since 2000.

ASSESSMENT OF HEALTH IMPACT OF PROPANE

A literature review for animal and or human studies that associated a particular level of propane with specific health effects was done. Data bases reviewed included but were not limited to the Centers for Disease Control (CDC), and The National Library of Medicine (NLM). The following exposure facts were discovered:

- Probable Routes of Human Exposure**:** Inhalation, skin and eye contact by liquid.[6]
- The most likely pathway by which the general public is exposed to propane is by inhalation due to the release of this substance from natural gas, natural gas food grills, and crude oil emissions. [7]
- Propane is a highly volatile compound and monitoring data indicates that it is a widely occurring atmospheric pollutant (SRC).[8]
- Studies in human volunteers showed that blood levels of propane could be detected after exposure to 250,000- 1, 000 ,000 ppb.[9]
- 8 adult volunteers were exposed to propane 250,000 – 1, 000,000 ppb for 1 min to 8hr over 1- 2 weeks. No abnormal physiological responses were observed in any volunteer. No cardiac abnormalities related to exposure were recorded[10]
- Humans exposed at 1,000,000 ppb (0.1%) propane for 10 minutes did not experience any CNS symptoms, while those exposed at 100,000, 000 ppb (10%) experienced distinct vertigo in 2 minutes. These data indicated that the onset of /CNS depression/ for propane **e**xposures occurred at a concentration between 1,000, 000 and 100,000, 000 ppb (eg, possibly at 47,000, 000 ppb as predicted by the model) and occurs quickly (under 15 minutes).[11]
- At concentrations up to 10% (100,000,000 ppb) propane caused no noticeable irritation to the eyes, nose, or respiratory tract.[12]

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[6] Sittig, M. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 1985. 2nd ed. Park Ridge, NJ: Noyes Data Corporation, 1985., p. 748] **PEER REVIEWED**

[7] [(1) NIOSH; NOES. National Occupational Exposure Survey conducted from 1981-1983. Estimated numbers of employees potentially exposed to specific agents by 2-digit standard industrial classification (SIC). Available at http://www.cdc.gov/noes/ as of Oct 24, 2006.] **PEER REVIEWED**

[8] [(1) Rappaport SM et al; Appl Ind Hyg 2: 148-54 (1987) (2) Kearney CA, Dunham DB; Am Ind Hyg Assoc J 47: 535-9 (1986) (3) Halder CA et al; Am Ind Hyg Assoc J 47: 164-72 (1986)] **PEER REVIEWED**

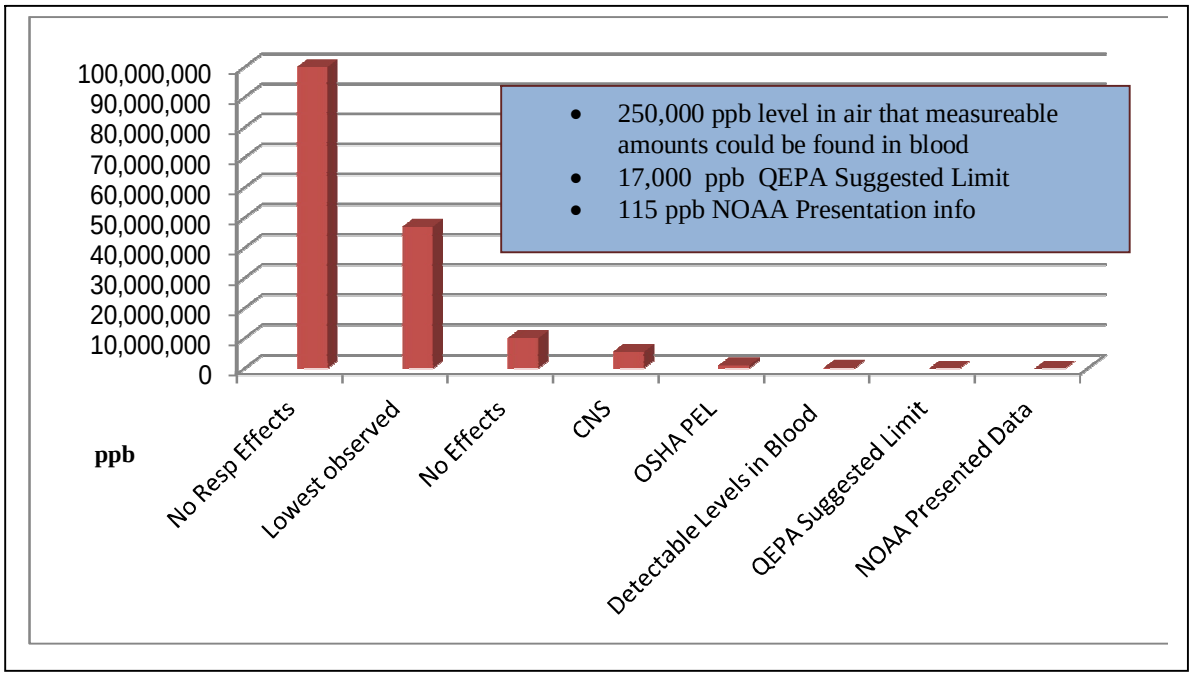
[9] [Snyder, R. (ed.) Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume 1: Hydrocarbons. Amsterdam - New York - Oxford: Elsevier, 1987., p. 263] **PEER REVIEWED**

[10] STEWART RD ET AL; US NTIS PB REP ISS PB-279205: 1-95 (1977)] **PEER REVIEWED**

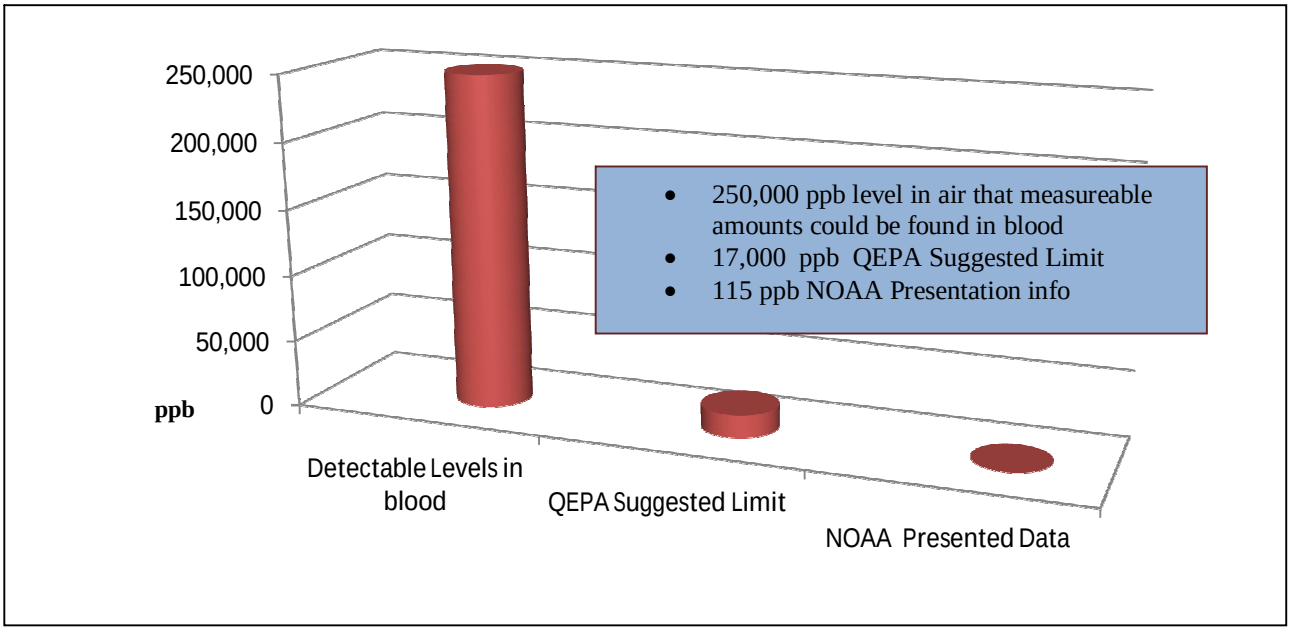
[11] [American Conference of Governmental Industrial Hygienists. Documentation of the TLV's and BEI's with Other World Wide Occupational Exposure Values. CD-ROM Cincinnati, OH 45240-1634 2005., p. 5] **PEER REVIEWED**

[12] Snyder, R. (ed.) Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume 1: Hydrocarbons. Amsterdam - New York - Oxford: Elsevier, 1987., p. 265] **PEER REVIEWED**

Graph A
Propane Concentrations and Health Effects



Graph B
Propane Concentrations and Health Effects



CONCLUSIONS

The Town of Erie understandably experienced a heightened awareness and concern surrounding levels of propane in the ambient air following a NOAA air monitoring presentation. QEPA was asked to conduct a scientific analysis of the data presented and to determine health effects (if any) that would result from exposures to the levels of propane presented.

Assessment of Health Impacts of NOAA Data

A review of peer reviewed literature from various official sources was done. The following was determined:

- Inhalation is the primary route of exposure for propane released.
- Regulatory guidelines for general human health such as a Minimal Risk Levels have not been created.
- An Occupational guideline of 1,000,000 ppb has been created by NIOSH, ACGIH, and OSHA for central nervous system impact. It is important to note that this level is for an occupational setting.
- The lower Explosive limit for propane is 23,000,000 ppb. Unless the gas is accumulating somewhere the levels of 115 ppb should not present an explosion hazard.
- Asthma is a concern of the citizens of Erie. Reviewed literature does not confirm these concerns. The County Health Department has reviewed the prevalence of asthma in the county and has not determined that asthma is a local issue of concern.
- Studies have shown that concentrations of up to 100,000,000 ppb propane **c**aused no noticeable irritation to the eyes, nose, or respiratory tract.[13] Propane at 115 ppb (the peak level found) does not present a health concern to the citizens of the town of Erie.

RECOMMENDATIONS

As evidenced in the Town of Erie and many similar settings, the lack of understanding of a problem can create the perception of an issue that can appear much worse than it really is. A scientific article written on this topic says the following. "Perceived *poisoning may manifest in numerous ways; however, all cases share certain characteristics. All are fostered by the wide availability of unreliable information about chemical safety, poor understanding of scientific principles and ineffective risk communication."*[14]. QEPA encourages the use of sound scientific data, the application of proper statistical principles to evaluate the data, the consideration of human perception, and clear messaging to help the public and decision makers in determining appropriate risk management decision.

REFERENCES

1. **2011 Air Chemistry Study at the Boulder Atmospheric Observatory**, Nitrogen, Aerosol Composition and Halogens on a Tall Tower February 16 – March 13, 2011, Brown, Steven S, NOAA Earth System Research laboratory Boulder, Colorado.

2. **2008 Hydrocarbon Emissions Characterization in the Colorado Front Range** – A Pilot Study, Petron, Gabrielle, Cooperative Institute for Research in Environmental Sciences, University of Colorado, Boulder, Colorado, USA 2 National Oceanic and Atmospheric Administration, Earth System Research Laboratory, Boulder, Colorado, USA

[13] Snyder, R. (ed.) Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume 1: Hydrocarbons. Amsterdam - New York - Oxford: Elsevier, 1987., p. 265] **PEER REVIEWED**

[14] Perceived Poison Kiristin A. Nan-agas, M.D. Mark A. Kirk, M Med Clin N Am 89 (2005) 1359 - 1378

3. **Interim Acute Exposure Guideline Levels** (AEGLS), National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (NAC/AEGL Committee) NAS COT Subcommittee for AEGLS 2009

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ADDENDUM 1

Federal/Health Guidelines & Glossary of Terms

FEDERAL GUIDELINES[15]

OSHA Standards: PEL for Propane 1,000,000 ppb

What is the Permissible Exposure Limit (PEL?

They are based on an 8 hour time weighted average exposure for 40 – 45 years. Enforceable limits provided by OSHA to protect workers against the health effects of exposure to hazardous substances. Table Z-1 8-hr Time Weighted Avg: 1,000,000 ppb (1800 mg/cu m). [29 CFR 1910.1000; U.S. National Archives and Records Administration's Electronic Code of Federal Regulations. Available from, as of August 30, 2006: http://www.gpoaccess.gov/ecfr **PEER REVIEWED**

American Conference of governmental industrial hygienist (ACGIH) :

TLV for Propane 1,000,000 ppb

What is the Threshold Limit Value (TLV)?

It is the term for airborne concentrations of a substance below which all workers are believed to be protected while exposed to it day after day for 8-hour period**s.** ACGIH classifies TLV in three ways

1) TLV-TWA: time weighted average concentration for a normal 8 hr work day or 40 hr work week

2) TLV-STEL short term exposure limit, or maximum concentration of a substance for

a) continuous 15 min exposure period

b) maximum of 4 such period per day

c) with at least one 60 minute exposure free period between two exposure periods and

d) provided the daily TLV-TWA is met

3) TLV-C Ceiling exposure limit or maximum exposure concentration that should not be exceeded under any circumstance while meeting the daily TLV-TWA 8 hr Time Weighted Avg *(TWA):* 1,000, 000 ppb. /Aliphatic hydrocarbon gases [C1-C4]/

[American Conference of Governmental Industrial Hygienists TLVs and BEIs. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati, OH, 2008, p. 11] **QC REVIEWED**

Excursion Limit Recommendation: Excursions in worker exposure levels may exceed 3 times the TLV-TWA for no more than a total of 30 minutes during a work day, and under no circumstances should they exceed 5 times the TLV-TWA, provided that the TLV-TWA is not exceeded. /Aliphatic hydrocarbon gases [C1-C4]/

[American Conference of Governmental Industrial Hygienists TLVs and BEIs. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati, OH, 2008, p. 5] **QC REVIEWED**

NIOSH Recommendations: REL Propane 1,000,000 ppb

What is a Recommended Exposure Limit (REL)?

It is an occupational exposure limit that is believed to be protective of worker safety and health over a working lifetime if used in combination with engineering and work practice controls, exposure and medical monitoring, posting and labeling of hazards, worker training and personal protective equipment.

Recommended Exposure Limit: 10 Hr Time-Weighted Avg: 1,000, 000 ppb (1800 mg/cu m).

[NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)] **PEER REVIEWED**

[15] Hazardous Substance Data Bank, National Library of Medicine, Toxnet. www.toxnet.nlm..nih.gov

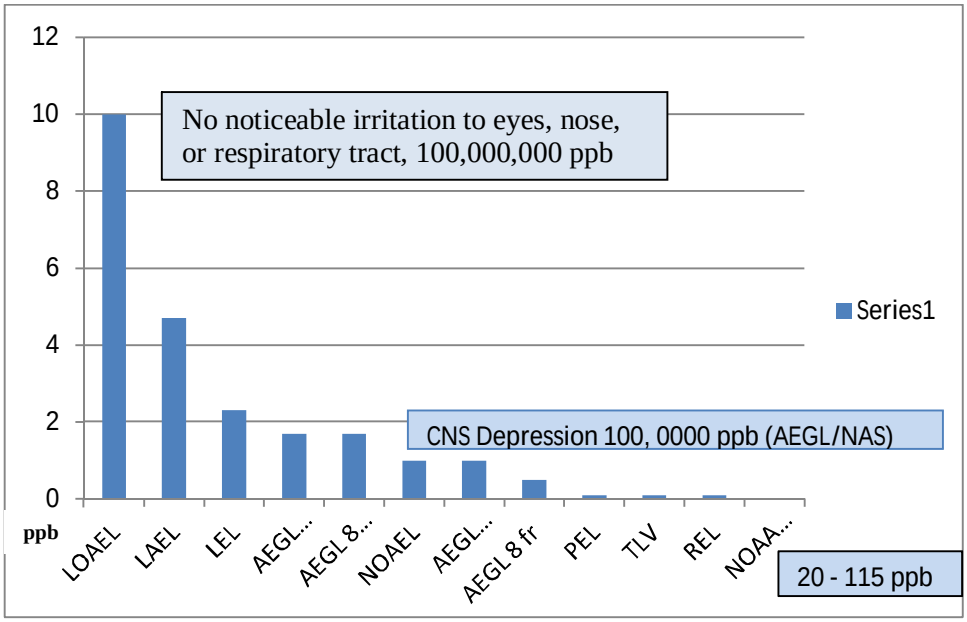
Immediately Dangerous to Life or Health: IDLH Propane 2,100,000 ppb

What is the Immediately Dangerous to Life or Health Guideline?

When exposure to airborne contaminants is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from such an environment.

Its purpose is to ensure that the worker can escape from a given contaminated environment in the event of failure of the respiratory protection equipment. The IDLH is considered a maximum level above which only a highly reliable breathing apparatus providing maximum worker protection is permitted. 2,100, 000 ppb [Based on 10% of the lower explosive limit for safety considerations even though the relevant toxicological data indicated that irreversible health effects or impairment of escape existed only at higher concentrations.]

[NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)] **PEER REVIEWED**



Health Guidelines For Propane Exposure

Level (ppb)	Designation	Effect occurring or being prevented	Source	Type	Duration
100,000,000	LOAEL Lowest Observed Adverse Effect Level	Vertigo	HSDB, NAS	Human	Acute
47,000,000	LAEL (modeled) Lowest Adverse Effect Level	Vertigo	HSDB	Human	Acute
23,000,000	Lower Explosive Limit	Explosion	NAS	Worker	Acute
17,000,000	AEGL-2 10 minutes AEGL-2 8-hours	Cardiac sensitization	NAS		Acute
10,000,000	NOAEL	Vertigo & CNS Depression	NAS		Acute
10,000,000	AEGL-1 10-minute	CNS Depression	NAS		Acute
5,500,000	AEGL-1 8-hour	CNS Depression	NAS		Acute
1,000,000	OSHA PEL 8-Hour Limit ACGIH TLV 8-Hour Limit NIOSH REL 10-Hour Limit	CNS Depression	OSHA ACGIH NIOSH	Worker	Chronic 45 yrs
17,000	QEPA's Estimate of an Suggested Ambient Air Limit for 24-hours per day	CNS Depression & Vertigo	**QEPA**	**Citizen**	**Chronic 70 yrs**
115	Maximum reported in NOAA 2011 Presentation		**Dr. Brown**		
20	Average in NOAA 2011 Presentation				

There are a lot of levels provided in the propane exposure, health guideline table and it is useful to differentiate them based on their purpose and merit.

The first is that there are *four types of data* in the table:

1. actual studies on exposure to animals or humans,
2. occupational exposure limits (PEL, TLV, REL),
3. emergency exposure limits (AEGLs), and
4. measured values at one location (BOA Tower) in Colorado.

Secondly, there are *different exposure duration periods* expressed in these data points.

- For the No Observed Effect Level (NOAEL) and Lowest Observed Effect Level (LOAEL) study results, these were the values selected from a variety of studies ranging from a one-time 2 minute exposure to 8 hours/day for 9 days over 2 weeks.
- For the occupational exposure limits (PEL, TLV, REL) it is either 8-hours/day or 10-hrs/day, 4 or 5-days/week for 40 to 45 working years.
- For the emergency exposure limits (AEGLs), they are one-time short-term exposures.
- For the BOA Tower it is X minutes for a peak value, and the average of a few weeks for the average value.

Third, there is the *exposed population*.

- In the case of the studies or the BOA Tower, *it is animals or humans*, and for the
- Occupational exposure limits it is healthy *young-middle aged adult humans* and for the
- Emergency limits it is the *general population*.

It can be shown that the exposure limits are set to prevent asphyxiation, central nervous system depression, cardiac sensitization and possible irritation (referred to as critical end points). In general, occupational exposure limits (OELs) are set by using either a NOAEL or LOAEL value and then reducing that value with a safety factor to account for: the length of the exposure, individual variability between people, variability between species, using an effect level (LOAEL) as opposed to a no effect level (NOAEL), severity of the endpoint (irritation versus cancer), and amount of data on and experience with the chemical. Safety factors range from 1 to 1,000 but are usually in the range of 3-300. Thus if one has a LOAEL of 1,000 ppb, then the exposure level might be 3 or 300. This is in contrast to ambient air limits for the general public where the very young, elderly and infirmed must also be protected, and because these exposures can be for 24 hours/day for 7 days/week for 70 years, versus occupational at 8-hours/day or 10-hrs/day, 4 or 5-days/week for 40 to 45 working years. As a result, safety factors for ambient exposure limits tend to range from 10 to 10,000 with most in the 30-3,000 range. As one can see from these safety factors, there is usually a 10-fold difference between occupational and general public exposure.

We analyzed propane to estimate an appropriate ambient exposure limit using this approach and consistent with others such as Calbrese's limit for Propene, a chemical with similar properties and effects[16]. We performed this using the LOAEL in the table and the NOAEL in the table using safety factors of 560 and 2,800, respectively. The results indicate an appropriate suggested ambient exposure limit of 16,800 to 17,900 ppb for 24 hours/day over a lifetime.

GLOSSARY OF TERMS

ATSDR	Agency for Toxic Substances and Disease Registry
CDC	Centers for Disease Control
CNS Depression	Central Nervous System Depression – a slowing of your body's central nervous system. It can slow your breathing and heartbeat, which can in turn result in coma or even death.
HSDB	Hazardous Substance Data Base
LOAEL	Lowest observed adverse effect level
NAS	National Academy of Sciences
NOAEL	No observed adverse effect level
NLM	National Library of Medicine
NIOSH	National Institute for Occupational Safety and Health
NOAA	National Oceanic Atmospheric Association
NOx	Nitrogen oxides
OSHA	Occupational Safety and Health Administration
ppb	Parts per billion
ppm	Parts per million
QA/QC	Quality Assurance/Quality Control
VOC	Volatile organic chemicals

[16] Dourson, Michael, et al: Differential Sensitivity of Children and Adults to Chemical Toxicity, II, Risk and Regulation. *Reg Toxicol Pharmacol* 35:448-467. 2002; EHC, Criteria, 239, Principles for Modeling Dose-Response for the Risk Assessment of Chemicals, 2009; Galer, DM, et al: Scientific and practical considerations for the development of OELs for chemical substances. *Reg Tox Pharmacol* 15(3):291-306. 1992; Haber, Lynne, and A. Maier: Scientific Criteria Used for the Development of Occupational Exposure Limits for Metals and Other Mining-Related Chemicals *Reg Toxicol Pharmacol*. 36:262-279. 2002; IPCS: Harmonization Project Document No. 2. Chemical-specific adjustment factors, 2005; Kalberlah, Fritz, et al: Uncertainty in toxicological risk assessment for non-carcinogenic health effects. *Regul Toxicol Pharmacol* 37:92-104. 2003; WHO: Guidance Document for the Use of Data in Development of Chemical-Specific Adjustment Factors, 2001; Calabrese, Edward, and Elaina Kenyon: *Air Toxics and Risk Assessment.* Lewis Publishers, Chelsea, MI. 1991; Whalley, David, et al: Regression method to estimate provisional TLV-WEEL-equivalents for non-carcinogens. *Ann Occ Hyg* 44(5):361-374. 2000; DeLorme, Mike, Amy Madl, Bob Sussman, and Tony Havics: "The Derivation of Occupational Exposure Limits", an 8 Hour Professional Development Course (PDC), at the *American Industrial Hygiene Conference & Exposition (AIHCE) 2011*, May 25-May 20, 2011, Portland, Oregon.

Addendum 2

Relevant Information & Relevant Abstracts

QEPA has provided this information for two reasons:

1. As a matter of convenience for the reader, and
2. To be transparent with respect to the data and sources that were reviewed and used to write this report.

RELEVANT ABSTRACTS

Source: Hazardous Substance Data Bank

Routes of Exposure

1. Inhalation, skin and eye contact by liquid.
   [Sittig, M. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 1985. 2nd ed. Park Ridge, NJ: Noyes Data Corporation, 1985., p. 748] **PEER REVIEWED**

2. NIOSH (NOES Survey 1981-1983) has statistically estimated that 2,071,479 workers (528,348 of these are female) are potentially exposed to propane in the US(1). Occupational exposure to propane may occur through inhalation and dermal contact with this compound at workplaces where propane is produced or used. Propane is widely detected in air(SRC). The most likely pathway by which the general public is exposed to propane is by inhalation due to the release of this substance from natural gas, natural gas food grills, and crude oil emissions. Monitoring data also indicate that the general population may be exposed to propane via ingestion of food and drinking water, although these pathways are considered minor when compared to inhalation(SRC).
   [(1) NIOSH; NOES. National Occupational Exposure Survey conducted from 1981-1983. Estimated numbers of employees potentially exposed to specific agents by 2-digit standard industrial classification (SIC). Available at http://www.cdc.gov/noes/ as of Oct 24, 2006.] **PEER REVIEWED**

3. The most probable route of human exposure to propane is by inhalation(SRC). Atmospheric workplace exposures have been documented (1-3). Propane is a highly volatile compound and monitoring data indicates that it is a widely occurring atmospheric pollutant(SRC).
   [(1) Rappaport SM et al; Appl Ind Hyg 2: 148-54 (1987) (2) Kearney CA, Dunham DB; Am Ind Hyg Assoc J 47: 535-9 (1986) (3) Halder CA et al; Am Ind Hyg Assoc J 47: 164-72 (1986)] **PEER REVIEWED**

4. Inhalation represents the major route by which propane is absorbed systemically. Studies ... in human volunteers showed that blood levels of propane could be detected after exposure to 250,000-1,000,000 ppb. ... Compared to respiratory absorption, dermal penetration of propane can be considered to be very low. ... the distribution /in tissues/ can be expected to follow the same pattern observed for butane.
   [Snyder, R. (ed.) Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume 1: Hydrocarbons. Amsterdam - New York - Oxford: Elsevier, 1987., p. 263] **PEER REVIEWED**

Toxicity Excerpts

1. HUMAN EXPOSURE STUDIES/ Eight adult volunteers of both sexes were exposed to isobutane, propane, or mixtures of the two gases (250,000 to 1,000,000 ppb for 1, 5, and 10 min and 1, 2, and 8 hr/day for 1 day or 2 wk) in a controlled environmental chamber for the purpose of monitoring their physiological responses. No untoward subjective responses were reported during or following these exposures. No abnormal physiological responses were observed in any volunteer. No cardiac abnormalities related to exposure were recorded. Serial computerized spirometric measurements revealed no pulmonary function abnormalities.
   [STEWART RD ET AL; US NTIS PB REP ISS PB-279205: 1-95 (1977)] **PEER REVIEWED**

2. HUMAN EXPOSURE STUDIES/ Human exposures to propane were consistent with the model predictions for /central nervous system depression/ onset and speed of action. Humans exposed at 1,000,000 ppb (0.1%) propane for 10 minutes did not experience any CNS symptoms, while those exposed at 100,000,000 ppb (10%) experienced distinct vertigo in 2 minutes. These data indicated that the onset of /CNS depression/ for propane exposures occurred at a concentration between 1,000,000 and 100,000, 000 ppb (eg, possibly at 47,000,000 ppb as predicted by the model) and occurs quickly (under 15 minutes).

[American Conference of Governmental Industrial Hygienists. Documentation of the TLV's and BEI's with Other World Wide Occupational Exposure Values. CD-ROM Cincinnati, OH 45240-1634 2005., p. 5] **PEER REVIEWED**

3. Skin, Eye and Respiratory Irritations:
   At concentrations up to 10% (100,000, 000 ppb) propane caused no noticeable irritation to the eyes, nose, or resp tract. [Snyder, R. (ed.) Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume 1: Hydrocarbons. Amsterdam - New York - Oxford: Elsevier, 1987., p. 265] **PEER REVIEWED**

Interactions

1. Propane, when used as an aerosol propellant with isobutane in deodorant and antiperspirant products (65 to 70% by wt), has not been shown to cause skin irritation in 125 human volunteers who applied the aerosol products twice daily for 12 wk.
   [Snyder, R. (ed.) Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume 1: Hydrocarbons. Amsterdam - New York - Oxford: Elsevier, 1987., p. 265] **PEER REVIEWED**

2. Physiological response to aerosol propellants.
   Stewart RD, Newton PE, Baretta ED, Herrmann AA, Forster HV, Soto RJ.
   Abstract
   Acute exposures to isobutane, propane, F-12, and F-11 in concentrations of 250,000, 500,000, or 1000,000 ppb for periods of 1 min to 8 hr did not produce any untoward physiological effects as determined by the methods employed which included serial EKG's and continuous monitoring of modified V5 by telemetry during exposure. Repetitive exposures to these four propellants were also without measurable untoward physiological effect with the exception of the eight male subjects repetitively exposed to 1000,000 ppb, F-11, who did show minor decrements in several of the cognitive tests. Of particular importance is the observation that none of the subjects showed any decrement in pulmonary function or alteration in cardiac rhythm as the result of exposure to concentrations of the gases or vapors far greater than encountered in the normal use of aerosol products in the home.

Surveillance

1. To describe patterns of inhalant abuse in New Zealand and discuss management... calls to the National Poisons Centre (NPC) from January 1, 2003 to December 31, 2004 were analysed. In addition, deaths following inhalational abuse were identified from the Institute of Environmental Science and Research Limited (ESR) database for 2001 and 2002 and available data for 2003. ... Seventy calls were classified as relating to inhalational abuse incidents. In abusers whose age was known, 83% were between 11 and 20 years, and 61% were male. Over half (44/70) of the calls involved abuse of propane or butane, either alone or in combination with a synthetic pyrethroid. ESR coronial data identified 11 inhalant abuse related deaths, most commonly attributed to cardiac effects. 73% of deaths were in teenagers and all but one fatality involved propane and/or butane. /The authors concluded that/ inhalant abuse is a persisting problem in New Zealand. NPC and ESR data demonstrate that teenagers are more likely to abuse inhalants than other age groups and butane and propane are the inhalants of choice. Acute management can be difficult, with significant mortality and morbidity. Continued education and other preventive measures are essential to help curb an extremely dangerous practice.
   [Beasley M et al; N Z Med J. 119 (1233): U1952 (2006)] **PEER REVIEWED**

RELEVANT ABSTRACTS

Source: NIOSH Health Hazard Evaluation

Carbon Monoxide (CO) Health Hazard Evaluation

Carbon monoxide is a colorless, odorless, tasteless gas produced by incomplete burning of carbon-containing materials. Major sources of exposure to CO are engine exhaust, tobacco smoke, and inadequately-ventilated combustion products from appliances and heaters that use natural gas, propane, kerosene, or similar fuels. On inhalation, CO acts as a metabolic asphyxiant, causing a decrease in the amount of oxygen delivered to the body tissues. CO combines with hemoglobin (the oxygen carrier in the blood) to form carboxyhemoglobin, which reduces the oxygen-carrying capacity of the blood. The initial symptoms of CO poisoning may include headache, dizziness, drowsiness, and nausea. These initial symptoms may advance to vomiting, loss of consciousness, and collapse if prolonged or high exposures are encountered. 33 F. Temperature and Relative Hum

HETA 91-254-2186 NIOSH INVESTIGATORS: *MARCH 1992 Calvin K. Cook ROPES & GRAY PHOTOCOPY CENTER Michael S. Crandall, CIH BOSTON, MASSACHUSETT*

RELEVANT ABSTRACTS - Asthma

Source: Medline

Asthma in children exposed to nitrogen dioxide in ice arenas.

Thunqvist P, Lilja G, Wickman M, Pershagen G.

Source: Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden. per.thunqvist@sachsska.sos.sll.se

Abstract

Very high concentrations of nitrogen dioxide (NO2) have been measured in arenas using combustion engine-powered resurfacing machines. This study was performed to compare the occurrence of asthma in children playing ice hockey in arenas using propane-powered machines and in children attending arenas using electric machines. Children regularly playing hockey in the arenas (nine propane, six electric) were sent a questionnaire, including questions on allergic disease and risk factors. Measurements of NO2 were performed with passive diffusion samplers during 3 consecutive days. The mean NO2 concentration in the propane arenas was 276 microg x m(-3) (range 28-1015 microg x m(-3)) and 11 microg x m(-3) (2-30) in the electric arenas. Questionnaires were answered by 1,536 children (78%), with an overall prevalence of asthma of 16%. The odds ratio (OR) for asthma was 0.9 (95% confidence interval (CI) 0.7-1.2) comparing propane arenas to electric. However, children in propane arenas with higher than median concentration of NO2 reported more wheezing (OR 1.4, 95% CI 1.0-1.9) and nasal symptoms (OR 1.7, 95% CI 1.3-2.3) than children in propane arenas with lower concentrations. In conclusion, children playing ice hockey in indoor arenas have a high prevalence of asthma, but it appears unlikely that increased exposure to combustion products, including nitrogen dioxide, is a major contributor to this excess risk.

PMID:12358342 [PubMed - indexed for MEDLINE] Free full text

Pollution & air quality Outdoor air quality

2011 Canadian Lung Association - Heating methods and open burning

The burning of fossil fuels such as coal, oil, natural gas, or propane (a refined component of oil or natural gas) as well as biomass sources such as wood, all release chemicals into the air. These chemicals include sulphur oxides (SOx), nitrogen oxides (NOx), volatile organic compounds (VOCS), particulate matter (PM), carbon monoxide (CO) and other toxic material. These pollutants can have direct effects on respiratory and cardiovascular health, and some of them cause acid rain or ground-level ozone, a principal component of smog. As well, these materials release carbon dioxide (CO2) which is a greenhouse gas and contributes to climate change. Generally speaking, with respect to fossil fuels and the release of pollutants, coal releases the most pollutants and natural gas is the cleanest choice.

RELEVANT ABSTRACTS
Source: Toxline National Library of Medicine

Health effects of selected chemicals 3. n-Propane

Authors: Berzins T

Source: Nord Vol:28 (1995) pp 175-92

Abstract:

N-**propane** is a colorless and odorless flammable gas which is used as fuel, as aerosol propellant and as refrigerant. An inhalation exposure to high doses, 47 000,000 to 55 000,000 ppb (85 to 99 mg/l), caused stupor in guinea pigs. n-**Propane** has also been shown to sensitize the heart to epinephrine-induced cardiac arrhythmias in mice and dogs. No reports on the lethality of n-**propane** in experimental animals have been located in the available literature. Very high doses of inhaled n-**propane** have been reported to be lethal to humans. The abuse of n-**propane** has increased among teenagers because of its capacity to cause euphoria. Repeated inhalation of n-**propane** has been reported to cause no pathophysiological signs in a patient after 6 months of daily inhalation. Symptoms on the central nervous system, such as headache and memory loss on the morning after exposure, were reported. No studies on the acute toxicity of n-**propane** by other administration routes were located in the available literature. In humans, liquid n-**propane** caused severe skin damage when accidentally dropped onto the skin. n-**Propane**-containing aerosols have been reported to cause similar effects when sprayed onto the skin. The injury, which occurs very rapidly, is like a deep frostbite with edema, hyperaemia and necrosis as symptoms. n-**Propane** containing mixtures (12 - 13% n-**propane**) have caused moderate irritation on the rabbit skin. No studies were found in the available literature on subacute, subchronic or chronic toxicity of n-**propane**. Two formulations containing 64.5$~ and 50% n-**propane**, respectively, were tested in a 90-day inhalation study in monkeys without any toxic symptoms detected. N-**propane** was shown to have no mutagenic activity in the Ames test with different strains of Salmonella typhimurium. No studies on carcinogenicity have been reported in experimental animals. Some incidences of associations between exposure to n-**propane** and melanoma of the skin have been noted. No further studies have been available. No studies concerning reproduction toxicity, teratogenicity, allergy and other immunotoxic effects of n-**propane** were located in the available literature. Following exposure to n-**propane** exhaust carbon monoxide poisonings have been reported. Several reports described neurologic symptoms in workers using n-**propane**-fuelled machines indoors. One case of death has been reported after use of a n-**propane** gas lamp in a closed station-wagon. Taken together, n-**propane** is a physically inert gas when used at low concentrations. Inhalation of n-**propane** at very high concentrations has been reported to have deadly outcome in humans. Exposure to lower concentrations caused different symptoms of neurologic character. Liquid n-**propane** has been reported to cause severe skin damage in humans.

RELEVANT ABSTRACTS

Propane and Greenhouse Gases

Source: Toxnet - National Library of Medicine

Propane Reduces Greenhouse Gas Emissions: A Comparative Analysis 2009.

Authors: Antes M. Brindle R. Kiuru K. Lloyd M,Munderville M

Author Address: Energetics, Inc., Columbia, MD.

Source: Govt Reports Announcements & Index (GRA&I), Issue 25, 2010

Abstract:

Sponsored by Propane Education and Research and Council, Washington, DC.

Growing concern about the potential effects of greenhouse gas (GHG) emissions has increased the focus on technologies and energy sources that can reduce these emissions. Policymakers in the United States and abroad are considering a variety of options for addressing the issue, including carbon cap-and-trade schemes, carbon taxes, and voluntary agreements to limit GHG emissions. As an Environmental Protection Agency (EPA)-approved clean alternative fuel, propane offers lower greenhouse gas emissions than many other energy options without compromising performance in a wide range of applications. This study quantifies the greenhouse gas emissions profile of propane compared to other energy sources in 13 selected applications of importance to the U.S. propane industry and the nation.

RELEVANT Abstract - Miscellaneous

Are Liquid Propane Gas Heaters a contributor towards Asthma together with other allergy symptoms ? Are Flued Heaters the only safe option ? This research explains the facts as well as the results may surprise you.

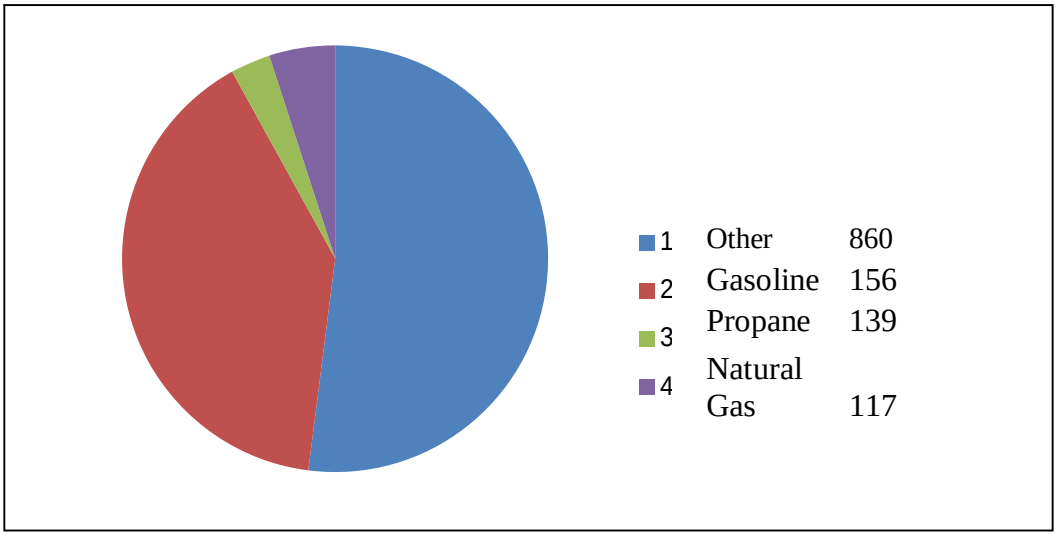
Jun 17, 2011 | Author: homegarden

Household propane gas heaters may be provoking asthma along with other breathing ailments and diseases in kids and the young and older simply because they discharge dangerous amounts of nitrogen dioxide, the writers of a new study have stated. The research of 388 Melbourne children aged 6 to eleven identified even small doses of nitrogen dioxide coming from natural gas heaters with out a flue - the tubing leading outdoors that permits by-products to leave - had been linked to substantial rises in uncomfortable throats, common colds, and absences from school and studies. Among the creators with the research, reported inside the International Magazine of Epidemiology, has cautioned it's apparent that the "level of some concern" of 300 models per hour suggested by the National Well being and Medical Research Council for indoor contact with nitrogen dioxide - over which complications with health may occur - is more than three times too much. Dr Louis Piloti shares knowledge in an article in today's Health-related Academic journal of Modern Australia that youngster's nicely becoming was impacted even when their own exposure to nitrogen dioxide was appropriately below the NHMRC-recommended maximum amount. Source: Gas Heaters The researchers stated that, provided the growing weight of investigation into the outcomes of nitrogen dioxide, emissions from unflued propane gas heating units and unflued natural gas stoves might function as the set off of consistent and unexplained asthmatic disease or unpredictable asthma. Other study have revealed nitrogen dioxide has effects on the lung's defense components, increasing the "permeability of bronchial mucosa to things that trigger allergies". But, the experts say, the mechanism for the way nitrogen dioxide has effects on the actual respiratory system plan is unknown. While the gas company has created unflued gas heating units with nitrogen dioxide emission ranges below suggested amounts, several outdated heaters have been nevertheless becoming made available and utilized in residences nowadays, the authors point out. Several heating units, especially these which are badly looked after, discharge nitrogen dioxide degrees of seven-hundred units per hour. It's a good technique to make certain that you have your current Gas Heating units examined for compliance regularly. Reference: Gas Heaters Earlier reports had demonstrated that high levels of nitrogen dioxide created by heaters also increased breathing

complications in younger asthma sufferers. A current analysis of ten non-smoking asthma sufferers, written and published in the international health related academic journal Lancet, found that nitrogen dioxide significantly enhanced their particular allergic reactions to typical asthma triggers such as home dust mites and the like. That harsh truth is the fact that flued natural gas heating units are generally safer than un-flued, most definitely if you are asthma suffering.

RELEVANT GRAPH

CO2 Formation [17]



Note

The amount of C02 produced when a fuel is burned is a function of the carbon content of the fuel. The higher the energy content the lower the C02 content. Propane is a relatively low producer of C02.

[17] Energy Information Administration

Background Information on Asthma

Source: Morbidity and Mortality Weekly Report (*MMWR*)

Current Asthma Prevalence — United States, 2006–2008

***Supplements*-Morbidity and Mortality Weekly Report (*MMWR*)**
January 14, 2011 / 60(01);84-86

Jeanne E. Moorman, MS[1] Hatice Zahran, MD[1] Benedict I. Truman, MD[2] Michael T. Molla, PhD[3]
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Asthma is a chronic inflammatory disorder of the airways characterized by episodic and reversible airflow obstruction, airway hyper-responsiveness, and underlying inflammation. Common asthma symptoms include wheezing, coughing, and shortness of breath (*1*). With correct treatment and avoidance of exposure to environmental allergens and irritants that are known to exacerbate asthma, the majority of persons who have asthma can expect optimal symptom control (*2*).

Multiple reports provide detailed surveillance information on asthma (*1*,*3*--*6*). A 1987 report that included asthma surveillance data for 1965--1984 identified differences among certain demographic groups by age, sex, and race/ethnicity (*3*). Subsequent asthma surveillance reports confirmed these differences and documented that the differences have persisted over time (*1*,*4*). These reports indicate that population-based asthma prevalence rates, emergency department visit rates, and hospitalization rates were higher among blacks than among whites, higher among females than among males, higher among children than among adults, and higher among males aged 0--17 years than among females in the same age group. In addition, more detailed analysis of ethnicity data demonstrated that different Hispanic groups had differing health outcomes. Among Hispanics, those of Puerto Rican descent (origin or ancestry) had higher asthma prevalence and death rates than other Hispanics (e.g., those of Mexican descent), non-Hispanic blacks, and non-Hispanic whites (*5*,*6*).

To examine whether disparities in asthma prevalence exist among certain demographic groups, CDC analyzed data from the National Health Interview Survey (NHIS) for 2006--2008. NHIS is an annual, in-person survey of the civilian, non-institutionalized U.S. population based on a multistage sampling of households (*7*). An adult family member is selected to act as a proxy respondent for children. NHIS routinely includes two questions that are used to estimate national asthma prevalence. The question, "Have you ever been told by a doctor or other health professional that you had asthma?" has been used as a lifetime prevalence measure for asthma since 1997. A second question, "Do you still have asthma?" was added in 2001 to assess current asthma prevalence. Consistent with previous CDC publications, respondents were considered to have current asthma if they answered "yes" to both questions (*1*,*4*). Race/ethnicity was categorized on the basis of the respondents' self-reported classification. Results for four racial/ethnic groups are reported: non-Hispanic white, non-Hispanic black, multiracial, and Hispanic of Puerto Rican descent. Current asthma prevalence also was estimated by sex (males and females), age group (children aged

0--17 years and adults aged ≥18 years), and federal poverty level. Analyses of disparities in disability status, education, geographic region, and other racial/ethnic populations were not included because of low prevalence or limitations due to data quality or manuscript length. Three years of survey data were combined to provide more stable estimates for relatively small groups. Analysis software accounted for complex sample design, and sample weights were used to produce national estimates. Estimates were age-adjusted by using the year 2000 age distribution, except those for children. Comparative terms used in this report (e.g., "higher" and "similar") indicate the results of statistical testing at $p<0.05$.

During 2006--2008, an estimated 7.8% of the U.S. population had current asthma (Table). Current asthma prevalence was higher among the multiracial (14.8%), Puerto Rican Hispanics (14.2%), and non-Hispanic blacks (9.5%) than among non-Hispanic whites (7.8%). Current asthma prevalence also was higher among children (9.3%) than among adults (7.3%), among females (8.6%) than among males (6.9%), and among the poor (11.2%) than among the near-poor (8.4%) and nonpoor (7.0%).*

When examined within the three federal poverty levels, prevalence by race/ethnicity was different than when race/ethnicity was examined alone. Among the poor, non-Hispanic whites and non-Hispanic blacks had similar prevalence (12.5% and 12.2%, respectively). In contrast, Puerto Rican Hispanics and the multiracial also had similar but substantially higher prevalence (22.4% and 20.5%, respectively). Among the near-poor, non-Hispanic blacks and non-Hispanic whites had similar prevalence (9.7% and 9.2%, respectively), and Puerto Rican Hispanics and the multiracial also had similar prevalence (14.9% and 13.6%, respectively). Among the nonpoor, non-Hispanic blacks had higher prevalence than non-Hispanic whites (8.4% and 7.0%, respectively). In contrast, the multiracial and Puerto Rican Hispanics had similar prevalence (13.4% and 10.4%, respectively).

For children (9.3% prevalence), current asthma prevalence was higher among Puerto Rican Hispanics (18.4%), non-Hispanic blacks (14.6%), and the multiracial (13.6%) than among non-Hispanic whites (8.2%). Asthma prevalence was higher among males (10.7%) than among females (7.8%). Among poor children, Puerto Rican children, multiracial children, and non-Hispanic black children had higher asthma prevalence (23.3%, 21.1%, and 15.8%, respectively) than poor non-Hispanic white children (10.1%) (Table).

For adults (7.3% prevalence), current asthma prevalence was higher among the multiracial (15.1%) and Puerto Rican Hispanics (12.8%) than among non-Hispanic blacks (7.8%) and non-Hispanic whites (7.7%). Asthma prevalence was higher among women (8.9%) than among men (5.5%). Among poor adults, Puerto Rican adults and multiracial adults had higher asthma prevalence (22.1% and 20.2%, respectively) than poor non-Hispanic black adults (10.9%) (Table).

For females of all ages (8.6% prevalence), current asthma prevalence was higher among the multiracial (17.4%), Puerto Rican Hispanics (16.9%), and non-Hispanic blacks (10.3%) than among non-Hispanic whites (8.7%). For males of all ages (6.9% prevalence), current asthma prevalence was higher among the multiracial (12.1%), Puerto Rican Hispanics (11.3%), and non-Hispanic blacks (8.5%) than among non-Hispanic whites (6.8%) (Table).

Because prevalence estimates for years before 2001 are not comparable to current definitions of asthma prevalence, only a limited number of years are available for trend analysis. The

prevalence differences between men and women, adults and children, non-Hispanic whites and non-Hispanic blacks, and poverty levels have not changed since 2001. The multiracial and Puerto Rican race/ethnicity groups are too small to produce reliable single-year estimates for assessing trends.

The results of this analysis are subject to at least four limitations. First, the asthma prevalence estimates in this report rely on self-report and are subject to recall bias. The respondent must correctly recall a physician diagnosis of asthma, which in turn requires that the physician diagnosis was correct and that the diagnosis was conveyed to the person. Because no definitive test exists for asthma, the diagnosis and self-report cannot be validated; however, a 1993 review of asthma questionnaires documented a mean sensitivity of 68% and a mean specificity of 94% when self-reported information on an asthma diagnosis was compared with a clinical diagnosis (*1*). Second, common to the majority of survey data, results might be biased because of response rates. NHIS is conducted by personal interview and had household response rates between 85% and 87% for the years included in this report. Third, because NHIS includes only the civilian, non-institutionalized population of the United States, results might not be representative of other populations. Finally, because NHIS is conducted only in English and Spanish, results might not be representative of households whose residents have other primary languages.

The findings of this report indicated that within the U.S. population, current asthma prevalence varied by multiple demographic and economic groups. Asthma was more prevalent among females, children, the poor, the multiracial, and Puerto Rican Hispanics. Findings from this report are comparable to those of previous reports (*1*,*3*,*4*).The exact cause of asthma is unknown, but health management strategies for asthma that take into consideration cultural and population-specific characteristics can reduce the occurrence and severity of asthma exacerbations (*8*).

Although the reasons for the disparities identified in this report are unclear, observed differences in asthma prevalence among certain demographic and socioeconomic groups (e.g., females, children, non-Hispanic blacks, Puerto Rican Hispanics, and the poor) might be indicators for underlying differences in genetic factors, higher levels of exposure to environmental irritants (e.g., tobacco smoke or air pollutants), and environmental allergens (e.g., house dust mites, cockroach particles, cat and dog dander, and mold). After asthma is diagnosed, heath-care access and actual use of the health- care system, financial resources, and social support are required to manage the disease effectively on a long-term basis (*8*--*10*). Research into the role of these factors among disproportionately affected demographic and socioeconomic groups can identify additional asthma control opportunities in these populations. Promoting targeted interventions that take into account cultural differences and population-specific characteristics can improve asthma management and subsequently reduce the asthma burden among disproportionally affected demographic and socioeconomic groups. For children, the use of multi-trigger, multi-component environmental interventions to improve symptom control and reduce missed days of school is recommended (*11*).